

Traumatic brain injury (TBI) is defined, by Traumatic Brain Injury Model Systems, as damage to brain tissue caused by an external mechanical force as evidenced by one of those: loss of consciousness due to brain trauma, post traumatic amnesia, skull fracture, or objective neurological findings that can be reasonably attributed to TBI on physical examination or mental status examination.

Many changes occur in the brain following a traumatic injury. Historically, the pathophysiology of TBI has been divided into primary and secondary injury.

Primary brain injury can result from a blow to the cranium or from rapid acceleration/deceleration, or rotation of the brain when it is slammed back and forth against the bony structures inside the skull. Primary brain injury can be further subdivided into focal and diffuse injury but most brain injuries have both of them. Focal injuries include skull fractures, subdural and epidural hematomas, intraparenchymal hemorrhages, cortical contusions and wounds:

- * Acute subdural hematoma results from tearing of the veins or cortical arteriole in acceleration/deceleration injuries. The prognosis is poor, and the mortality is high because of the severity of the underlying parenchymal injury.

- * Epidural hematoma results from direct trauma to the skull, which results in laceration of the dural veins and arteries.

- * Intraparenchymal hemorrhages are areas of hemorrhage within the brain parenchyma. They can be secondary to the injury by bone fracture but also can occur from shear strain and rotational forces. They occur mostly in the frontal and temporal lobes, but may be found in the cerebellum and brain stem. DAI can be found scattered throughout the brain and brainstem but is more common in the deeper cerebral white matter, corpus callosum, and cerebral peduncles. In severe DAI, the axon disconnects from the neuronal soma within 6–12 hours. The distal segment of the axon degenerates and there is a deafferentation and subsequent degeneration of the target area.

- * Cortical contusions may extend through the cortex and into the subcortical white matter. The distribution of cortical contusions is predictable due to the bony protrusions that the brain strikes along the interior wall of the skull. The movement of the brain in the skull, caused by acceleration–deceleration and rotational forces, causes certain cortical areas to be more susceptible to injury. The most frequently injured brain areas are: frontobasal lobe and anterior/lateral temporal regions.

The most recognized type of diffuse injury is diffuse axonal injury (DAI). DAI refers to extensive damage to the white matter of the brain. There is shearing of the white matter with widespread mechanical disruption of axons and myelin sheaths in the cerebral hemispheres, corpus callosum, brain stem, and

cerebellar peduncles. Usually, there is a deep and prolonged coma from the moment of impact. This sort of injury is related with high-speed acceleration-deceleration injuries, such as those caused by motor vehicle collisions.

Secondary brain injury. This lesion occurs in the postinjury phase and is due to associated physiological impairments resulting from the primary injury. Decreased cerebral perfusion, increased intracranial pressure, brain edema, hypoxemia, and circulatory shock (hypotension) may increase brain injury. The main goal in the acute phase after a TBI is to stop the oxygen deprivation and increase the blood flow to maintain cerebral perfusion pressure. Most secondary injury occurs during the first 12–24 hours after trauma, but it may occur at any time during the first 5–10 days in patients with very severe primary brain injury (Cooper, 1985). Focal ischemia, which occurs in approximately 90 percent of TBI cases, is implicated in initiating many of the toxic neurochemical processes. Increased levels of excitatory amino acids, endogenous opioid peptides, and acetylcholine are among the neurochemical agents that contribute to secondary brain damage.

Elevation of intracranial pressure are related with poorer functional outcomes, especially if pressure is beyond 40 mmHg. Cerebral edema or swelling is attributed to disruption of the blood-brain barrier and impairment of vasomotor autoregulation with concomitant dilatation of cerebral blood vessels.

Physicians can not treat primary brain injury but they can act decreasing secondary brain injury as a way to avoid further brain injury.